

can do." This is just the point of the whole problem about which I differ emphatically with Prof. Mills, for with her present limited training and destroyed initiative the nurse has not the training she should have in executive work and in the vast majority of hospitals no social work whatever.

We cannot soberly contemplate the huge health problems of the war and of the later readjustment in the light of what we can see now, and not tremble at the risk we run in not preparing more than we are doing to meet them.

Original Articles

THE INTERRELATIONSHIP OF ASTHMA AND TUBERCULOSIS.*

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Asthma as a disease entity does not exist. Among other things it may be an anaphylactic manifestation. This aspect of the subject has been very fully and ably investigated by Talbot and Goodale of Boston and Selfridge, the latter having made a particular study of the polius and flora of California and the Pacific States. It may be due to chronic infection in some other portion of the body; it may be the result of a chronic congestion and inflammation as exists in cases of prolonged cardiac insufficiency. The particular type of asthma that is associated with pulmonary tuberculosis also bears out the idea that asthma, asthmatic breathing, is a symptom and not a disease in itself. Consequently, let me here explain what I mean by asthma.

To me it means a condition symptomatically consisting in more or less prolonged expiration and sometimes inspiration, rather constant for days, weeks or months, associated with exacerbations of more intense discomfort and often increased by exertion. To the patient himself, it may be represented only by a rattling in his "upper air passages" and a slight dyspnea, especially with expirations. The physical signs consist in prolonged expiration, general or localized, accompanied by musical or sonorous rales. With cough or increased depth of respiration, these signs are often increased.

Many older writers on the subject were quite unanimous that these two diseases were antagonistic and seldom existed together. Brügelmann,¹ Sarda and Vires,² Grasset,³ Rancoule⁴ and Baur⁵ held that asthma was antagonistic to tuberculosis and never secondary to it, but always primary. Landouzy⁶ suggested that asthma might be an anaphylactic reaction to the tubercle bacillus. Roboule⁷ referred to a pretubercular asthma, mean-

ing that there are individuals with a nearly inactive tuberculosis manifesting itself by producing asthma. Osler⁸ observed that one of the early signs of tuberculosis might be asthma with its wheezing and sibilant rales. Socca,⁹ from the study of 840 cases of asthma, concluded that nearly all were due to tuberculosis. Reynier,¹⁰ on the contrary, in an article of considerable length, concludes that asthma is in no manner dependent on tuberculosis. Hoffman¹¹ believes that if they do exist in the same individual each gives up its more pronounced symptoms and the asthmatic attacks become less marked than in other individuals and the tuberculosis becomes a fibroid phthisis. Giffin¹² in reviewing 83 cases of asthma in the St. Mary's Hospital records found only three cases of definite tuberculosis but expressed a suspicion of there being a possible latent tuberculosis as a basis in others.

That enlarged bronchial lymph glands, which may be accepted as an evidence of tuberculosis, were found in asthmatics was first reported in a series of eighteen cases by Chelmonski¹³ in 1912. Lawrason Brown¹⁴ is of the opinion that cases of chronic tuberculosis not infrequently developed asthma. Last year Orville Brown¹⁵ published a book on asthma and in it he puts forth a theory—"The non-passive expiratory theory"—to explain the morbid condition which exists at the time of asthmatic attacks, whether mild or severe. His theory is that with ordinary respiration there is an influx of blood and lymph toward the periphery of the lung during inspiration and a retardation of the flow during expiration due to the increased intra alveolar pressure; also that by the act of frequent coughing, sneezing, etc., a still greater increase of intra alveolar tension is created as compared with the pressure in the bronchi and there is a greater impediment placed in the way of blood in the bronchial veins and of the lymph. This causes an obstruction of varying degree in the larger bronchioles with expiration and it gives the sibilant and musical nature to the breathing and rales. There is much more to the theory but this is all that need be considered here.

This requires at least two predisposing factors—paroxysms of coughing and congestion or inflammation in the moderate sized bronchioles. Let me emphasize the fact that asthma is not always a diffuse process but often limited to a small area in one lung. Although the signs may be localized the symptoms may be localized or general. In tuberculosis, we have both of these factors. In order that we may understand why asthma occurs in one or another part of the lungs, may I briefly

* Read before the Forty-seventh Annual Meeting of the Medical Society of the State of California, Del Monte, April, 1918.

1. Brügelmann, W.—*Therap. Monatsh.*, 1898, xli, p. 320; *Das Asthma*, Wiesbaden, 1901, p. 39.
2. Sarda, G. and Vires, J.—*Revue de la Tuberculose*, 1894, ii, p. 121.
3. Grasset—*Leçons Cliniques*, 1896, from Reynier (No. 21).
4. Rancoule—*Thèse de Montpellier*, 1899, from Reynier (No. 21).
5. Baur—*Forme Clinique de la Tuberculose pulmonaire*, from Reynier (No. 21).
6. Landouzy, M.—*Presse méd.*, 1912, p. 892.
7. Roboule—*Thèse de Montpellier*, 1904, from Reynier (No. 21).

8. Osler, Wm.—*Principles and Practice of Medicine*, 1905, Ed. 6, p. 322.

9. Socca, F.—*Arch. gén. de méd.*, 1906, cxcvii, p. 1601.

10. Ibid., 1907, cxcviii, p. 353.

11. Reynier, Leopold de—*Proc. Sixth Internat. Cong. on Tubercs.*, 1908, i, p. 1133.

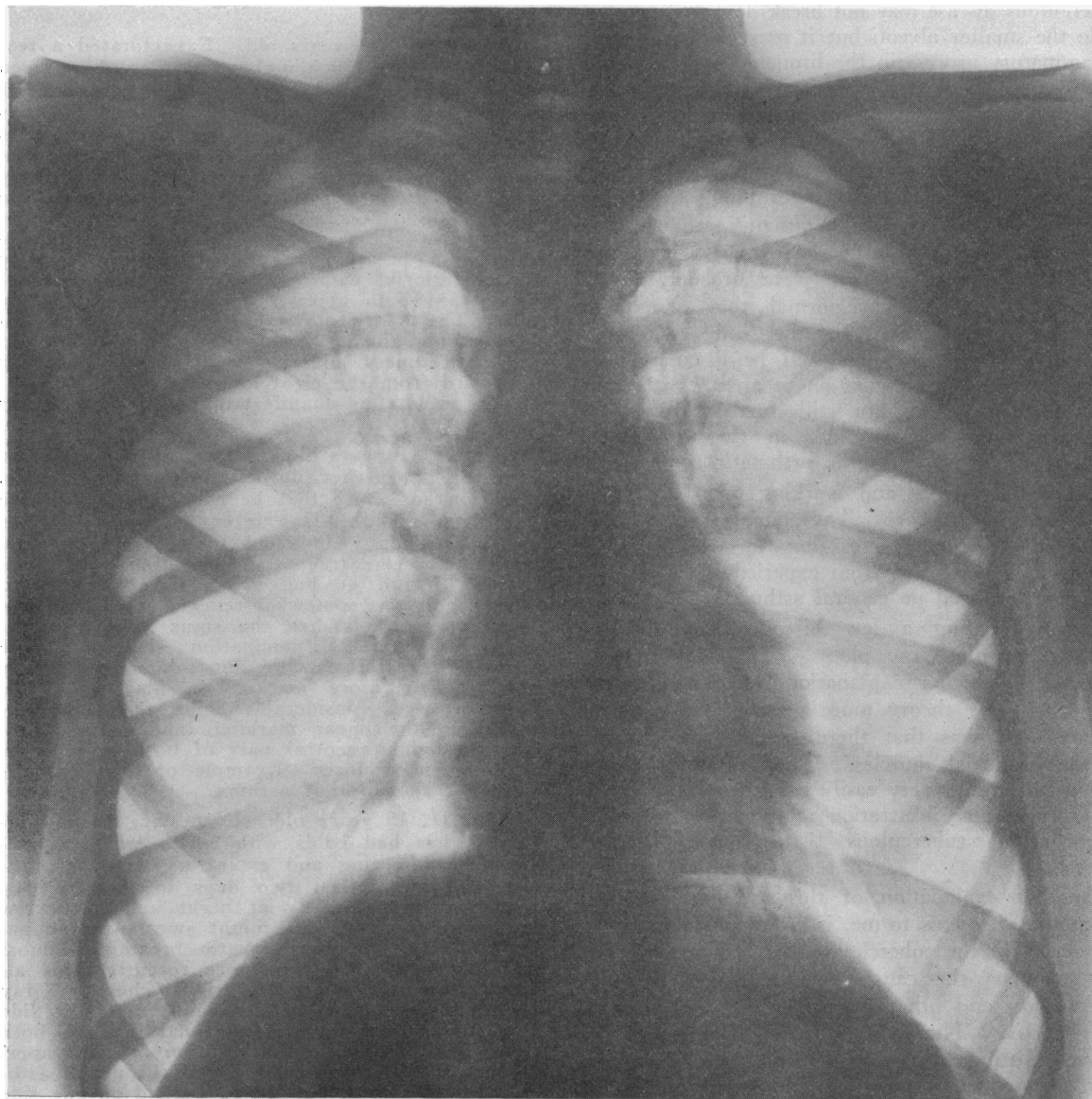
12. Hoffmann, Frederick A.—*Nothnagel's Encyclopedia of Practical Medicine*, 1902; *Bronchi, Lungs and Pleura*, p. 219.

13. Giffin, H. Z.—*Am. Jour. Med. Sc.*, 1911, cxlii, p. 869.

14. Chelmonski, Adam—*Deutsch. Arch. f. Clin. Med.*, 1912, cv, p. 522.

15. Brown, Lawrason—*Osler and McCrae, Modern Medicine*, Lea & Febiger, 1913, Ed. 2, i, chap. vi, p. 376.

16. Brown, Orville—*Asthma*, C. V. Mosby Co., 1917, p. 179.



Case No. 3. Example asthma more marked on right side.

review the main points in the development of tuberculosis.

Whether primarily or secondarily infected the first evidence of pulmonary tuberculosis is an enlargement of the bronchial glands. Their enlargement is a part of the normal mechanism for the battle against the disease. It is through their functioning that antibodies are manufactured. When the individual is enjoying good health we do not interpret the glands as a tubercular focus; it is only when the person is below normal that we point to these as of potential or possibly actual danger. In this latter type of individual the glands are swollen and more actively diseased. Their presence irritates the adjoining bronchi, setting up a low grade inflammation which may become the seat of secondary infection. So here we have the factors above stated which may start the vicious circle for an asthmatic condition. This

condition may occur during childhood or later life and may irritate one bronchus and hence one lobe, or one lung or both lungs. This is a very important factor in properly interpreting *localized* asthmatic breathing. From the glandular condition slowly or rapidly develops a peribronchial fibrous thickening extending outward toward the periphery. This may involve one lobe much more than another. All this time the individual is manufacturing antibodies, and if these are sufficient the disease remains confined and not actually dangerous. If the scale turns against the individual in this struggle, we find a breaking down of the glandular or lymphatic elements at the hilus or nearer the periphery and a focus started in the lung tissue. Again depending upon the location and also upon the extent of the process, we may have one or more lobes involved by an irritating process in a bronchiole and a factor

established for a possible localized asthma. The tuberculous disease may not break down so as to invade the smaller alveoli but it may cause a chronic inflammatory process in the bronchioles on which is developed a secondary infection. The possible tuberculous etiology for this secondary infection must not be overlooked in such instances.

The pulmonary markings as seen in the X-ray are of great importance in determining this point. Dunham, Boardman and Wolman¹⁶ studied these linear markings and hilus shadows and concluded that where these shadows were broader, denser and more beaded than normal they should be considered tubercular. In the slides which I will show these markings will be discussed. Of course, as the disease goes on to areas of caseation and softening there are still further causes for the congestion and swelling in the bronchioles, thus paving the way for an asthmatic condition.

From this survey and working upon the "non-passive expiratory theory," we have explained clinically and pathologically that localized foci of tuberculosis may, and from experience do, actually set up a localized or general asthma.

Before reading a few histories and showing some slides of X-ray plates, we should at least consider two other explanations of this asthmatic dyspnea. The theory more generally accepted in recent years was that there was set up a spasm of the bronchial muscles. The irritation causing this spasm, could very easily come from this latent or pretubercular infiltration at the hilus or in the peribronchial tuberculous tissue toward certain lobes.

Another explanation of this asthma, and least acceptable, it seems to me, is an anaphylactic status the result of the tubercle bacilli. In certain cases this may be the cause, for we know that the streptococci and other organisms are the cause of anaphylaxis. There are phases of more or less active splitting of the bacilli and their absorption and, consequently, periods of exacerbation of asthmatic dyspnea.

The localized areas of asthmatic breathing, I wish to emphasize again in conclusion. When we find persistent or repeated localized asthmatic breathing and musical rales in a lobe or lung, we should keep in mind the fact that there is probably a mechanical irritation and then inflammatory cause for it, and this cause is often tuberculosis in a very early or more advanced condition. It may be of considerable benefit in the early recognition of tuberculous disease. This has impressed me in several of the drafted men that I have had occasion to examine. The X-ray has shown definitely beginning tuberculous lesions. We should not be misled in our diagnosis by temporary disappearance of signs and symptoms for as the activity and intensity of the tuberculous area increases or di-

minishes, there will be an increase or diminution of signs.

Miss M. T., 20 years old. Expectored a teaspoonful of blood last night, and there have been streaks of blood in the sputum at the time of her menstruation for the last three months. She has had "bronchial trouble" for the last year. This has consisted in wheezing and expiratory dyspnea, especially while lying down or when particularly tired. At times this has been quite severe. She has had slight fever at times and has lost about 10 pounds. Physical examination shows markedly harsh breathing at both hiluses anteriorly and posteriorly, with increased harshness extending toward both apices, the left more than the right. The X-ray shows definitely enlarged bronchial glands and a fine infiltration extending up into both apices and a few tubercles extending downward from the right lower bronchus. Example of asthma as manifestation of pretubercular state.

Robert B., 9 years old. Attacks of asthma twice a month or oftener during the last two years. Condition so severe that he has lost considerable time from school. Has fever at times and has lost 13 pounds. Had had tonsils removed and four teeth were extracted as possible foci of infection. Although he had a moderate reaction to oatmeal and was somewhat relieved when this was discontinued in his diet, the signs remained very much the same. Examination showed diffuse asthmatic breathing with musical and sonorous rales slightly more marked on his right side. X-ray showed considerable hilus thickening increases in the linear markings and diffuse mottling throughout central part of both lung fields. Probable tuberculosis. Example of glandular tuberculosis as cause of asthma.

Philip D., 15 years old. Ever since he can remember has had colds with cough which is followed by wheezing and expiratory dyspnea. Attacks of asthma last two days to three weeks. Often perspires slightly at night and on a few occasions has had severe night sweats. Examination at first showed asthmatic breathing almost equal on both sides but more recently it is almost entirely confined to right chest. X-ray shows increase in hilus thickening on right side with slight thickening of the markings to both uppers with some slight infiltration of right apex. Suggestive tuberculosis as basis of bronchial asthma. Example of asthma more marked on one side than the other with X-ray confirmation.

Mr. N. T., age 46. Operated a dry drill in a gold mine six years, and since this time pick and shovel work. Had a slight hacking cough a good deal of this time but never paid any attention to it. Eleven months ago became much worse, following pleurisy, and sputum become purulent. For the last six or seven months has had almost constant and very distressing expiratory dyspnea. He volunteers the information that it comes entirely from the left side. Examination shows marked wheezing on his left side, almost none on his right. Harsher breathing at his left apex with many consonating rales. X-ray shows diffuse pneumoconiosis with denser infiltration at the left apex with probable cavity formation. Sputum positive for tuberculosis. Example of asthma complicating tuberculosis (one sided) implanted on pneumoconiosis.

Miss C. K., age 30. Attacks of asthma for 15 years with two years as the longest period of freedom. Eight and four years ago hemoptysis of one dram and one ounce. During that time lost from 125 pounds to 84 pounds. Now weighs 103. At present slight wheezing but no dyspnea. Has had every possible focus of infection searched and removed when found. Examination showed

16. Dunham, H. K., Boardman, W. W., Wolman, S.—Stereoscope X-Ray Examinations of the Chest with special reference to the diagnosis of Pulmonary Tuberculosis. Johns Hopkins Hospital Bulletin, vol. xxii, No. 245, July, 1911.

active tuberculosis at both apices with signs of cavity at the right apex. Elsewhere in the chest there was asthmatic breathing and musical rales. X-ray showed moderate infiltration both apices with a small cavity at the right apex. Asthma preceding active tuberculosis.

Miss N. D., age 24. Asthma as a child and up till ten years old. Then removed from sea coast and gradually during next five years it disappeared. Free from it for eight years till one year ago when it reappeared. Ten months ago severe pleurisy. Eight months ago hemoptysis of one pint. Wheezing and dyspnea very marked at this time. Less at present. Examination showed active tuberculosis at both apices with definitely prolonged expiration and musical rales in left upper lobe. X-ray shows marked infiltration in both apices with cavity at the apex. Example of asthma as evidence of probable bronchial tuberculosis as a child and its recurrence when the disease was again becoming active.

Mr. A. P., age 25. Periodic attacks of asthma during the last 14 years, more often at the end of the week. Last three to four days and length of entire freedom varies from one to three months. Every possible focus of infection has been investigated and whenever trouble was found it was removed, without as yet benefiting the asthma. Examination showed wheezing throughout both chests and scattered musical rales. X-ray showed marked hilus and peribronchial infiltration with definite dense areas in both apices. These seem definitely tubercular to me. Example of frequent asthmatic attacks and evidence of apical tuberculosis.

Mrs. O. Five years ago asthma and tuberculosis were both discovered. At this time asthma was not severe but two years ago it became very disturbing. Since coming to Arequipa Sanatorium only has occasional wheezing spells. Examination has always shown prolonged expiration and musical rales, very much more marked on the right side than on the left. There are signs of cavity at the right apex and more often consonating rales on the right side than on the left. X-ray shows a moderate infiltration on the left side with a more marked infiltration on the right, especially in the upper and middle lobes, with a large cavity at the right apex. Example of persistent asthmatic breathing confined to the side of chief involvement.

Miss C. C. As a child always had wheezing with colds, but no difficulty in breathing. Two and a half years ago grippe, which led to the discovery of tuberculosis. For last 14 months wheezing only occasionally. If severe the wheezing seems to bring on coughing spells and with this a ropy expectoration. Examination has always shown more prolonged expiration and musical rales in her right middle and lower lobes than elsewhere. Sputum has been positive for tuberculosis. X-ray shows chiefly a fibrous tuberculosis and more marked in the right middle and lower lobes. Example of localized persistent asthmatic breathing in a tuberculous individual.

Mrs. A. Perfectly well till one year ago, when asthma began. This was very genuine, with difficult and prolonged expiration and ropy expectoration. Six months ago this was helped by some salty medicine, but at the same time she began to have fever, sweats and purulent expectoration. Examination showed active signs of consolidation and cavity on the left and a small amount of latent activity on the right. X-ray confirmed these findings. Pneumothorax was artificially induced and she has had no asthma since. Her tuberculosis is also very much improved.

Tuberculin is indicated in certain cases where there is chiefly a latent tuberculosis. Pneumothorax may be used where the disease is more active and confined to one side. Potassium iodide, at least theoretically, is contraindicated because of its possible action of breaking down tuberculous tissue.

THE VALUE OF RENAL FUNCTIONAL STUDIES IN THE PROGNOSIS AND TREATMENT OF NEPHRITIS.*

WITH SPECIAL REFERENCE TO THE RENAL TEST DIET.

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INTRODUCTORY.

No addition to our knowledge of any group of diseases can be without significance for prognosis and treatment. One need only glance at current medical periodicals to be convinced of the time and effort which is now being given to the study of kidney function. So much work has surely, by this time, produced some results which the practitioner of medicine can use for the benefit of his patients. General practitioners wish to know those tests which are of practical value and those which are of academic interest. The number of tests has multiplied so rapidly within recent years that considerable uncertainty concerning their mode of application and their interpretation prevails in the minds of those who would employ them. Functional tests will become more generally used and hence of more value when we know which ones can be discarded without loss, and which combination of tests (the smaller, the better) will yield all the information necessary in any given type of disease.

In interpreting functional tests physicians have had a tendency to make the judgment of laboratory findings too precise, and to place too much emphasis upon the knowledge gained from them. Such a tendency will go far to make the physician mechanical in the interpretation of the facts laid before him and to lead him astray. It has become clear to those who with care are in the habit of correlating laboratory with clinical observations that no functional test can be used as direct evidence for the diagnosis of the anatomic or etiologic type of disease present, but only indirectly as an assistance to the experienced in arriving at a final judgment. While a correct anatomical diagnosis is always desirable, it is of no especial importance in determining prognosis and treatment. What is of practical value to the general practitioner is a knowledge of the kind and degree of functional damage.

METHODS OF TESTING KIDNEY FUNCTION.

During the past few years, renal functional capacity has been studied chiefly in one of two ways: first, by testing the excretory capacity through the quantitative determination of the eliminative power of the kidneys for various substances in the urine, either foreign substances like phenolsulphonephthalein or the normal urinary constituents,—water, salt, and nitrogen; second, tests of retention through quantitative determination of the concentration of certain substances in the blood, either non-protein nitrogen as a whole or one of the

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